In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: January 21, 2020

PAUL DROBBIN,	*	No. 14-225V
Petitioner,	*	Special Master Sanders
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Ruling; Influenza ("flu") Vaccine; Sensory Polyneuropathy; Small Fiber Neuropathy; Motor Neuropathy.
Respondent.	*	

Martin Jeffrey Rubenstein, Martin Rubenstein, Staten Island, NY, for Petitioner.
Lisa Ann Watts, United States Department of Justice, Washington, D.C., for Respondent.

RULING ON ENTITLEMENT[1]

On March 24, 2014, Paul Drobbin ("Petitioner") filed a petition for compensation pursuant to the National Vaccine Injury Compensation Program.[2] Petitioner initially alleged that the influenza ("flu") vaccine he received on November 18, 2011, caused him to suffer from fever; reactive arthritis/joint pain; muscle fatiguability; muscle spasms; extreme fatigue; tingling and numbness; fasciculations and asymmetric muscle atrophy that started in his calves and hands, forearms, and affecting his back and leg muscles, buttocks, and shoulders; neuropathy; exercise intolerance; breathing problems; and declining lung function. Pet. at 1, ECF No. 1. Petitioner ultimately narrowed his alleged injuries to "combined sensory and motor polyneuropathy, overlapping with neuromuscular juncture[3] dysfunction." Pet'r's Pre-Hr'g Br. at 1, ECF No. 76.

[1] This Ruling shall be posted on the website of the United States Court of Federal Claims, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, § 205, 116 Stat. 2899, 2913 (codified as amended at 44 U.S.C. § 3501 note (2012)). **This means the Ruling will be available to anyone with access to the Internet.** As provided by Vaccine Rule 18(b), each party has 14 days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b).

[2] The Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C. §§ 300aa-10 et seq. (hereinafter "Vaccine Act," "the Act," or "the Program").

[3] Although Petitioner alleges neuromuscular juncture dysfunction, there were no references to such a condition found aside from the petition and Dr. Brawer's expert report. Dr. Brawer did not testify to clarify. I will refer to this condition throughout this decision as neuromuscular junction dysfunction, which has previously been asserted as an injury in the Program. *See generally D.G. v. Sec'y of Health & Human Servs.*, No. 11-577V, 2019 WL

After carefully analyzing and weighing all the evidence and testimony presented in this case in accordance with the applicable legal standards, I find that Petitioner has provided preponderant evidence that the flu vaccination he received on November 18, 2011, was a substantial factor in the development of his small fiber neuropathy. Respondent has failed to rebut Petitioner's claim by establishing an alternative cause with preponderant evidence. Accordingly, the case shall proceed to damages.

I. Procedural History

Petitioner filed his petition on March 24, 2014. Pet. Over the next eleven months, Petitioner filed forty-two medical records and a compact disc containing additional records. Pet'r's Exs. 1–12, Notice Compact Disc Received, docketed June 10, 2014; Pet'r's Exs. 13–39, ECF Nos. 11-1–11-7, 12-1–12-3, 13-1–13-2, 15–16, 18, 20-1–20-6, 23-1–23-15, 25-1–25-5. Petitioner filed his statement of completion on February 25, 2015. ECF No. 27.

On May 6, 2015, Respondent filed his Rule 4(c) report. Resp't's Report, ECF No. 29. In it, Respondent argued that Petitioner's records do not support his alleged diagnosis, and Petitioner's neurologic symptoms have been attributed to B6 toxicity, Lyme disease, and CPT II deficiency. *Id.* at 19.

On July 22, 2015, Petitioner filed an expert report by Arthur E. Brawer, M.D., three pieces of medical literature, Dr. Brawer's C.V., and four medical records. Pet'r's Exs. 40–48, ECF Nos. 31-1–31-9. Petitioner filed fourteen additional medical records on November 9, 2015. Pet'r's Exs. 49–62, ECF Nos. 34-1–34-14. A month later, Respondent filed his responsive expert report by Peter Donofrio, M.D., Dr. Donofrio's C.V., and four pieces of medical literature. Resp't's Exs. A–F, ECF Nos. 35-1–35-6. Petitioner filed additional medical records on December 28, 2015, and March 2, 2016. Pet'r's Exs. 63–66, ECF Nos. 36-1–36-4; Pet'r's Exs. 67–68, ECF Nos. 40-1–40-2.

On March 18, 2016, Petitioner filed his first supplemental expert report by Dr. Brawer. Pet'r's Second Expert Report,[4] ECF No. 41. Ten days later, Petitioner filed an additional medical record. Pet'r's Ex. 69, ECF No. 42. On June 1, 2016, Respondent filed his first supplemental expert report by Dr. Donofrio. Resp't's Ex. G, ECF No. 46.

From July 6, 2016 to June 8, 2017, Petitioner filed five more supplemental expert reports, three authored by Dr. Brawer and two authored by Allan Earl Rubenstein, M.D. Pet'r's Ex. 71, ECF No. 53-1; Pet'r's Exs. 91, 93, ECF Nos. 58-2, 58-4; Pet'r's Ex. 96–97, ECF Nos. 60–61. Along with Petitioner's supplemental expert reports, Petitioner filed five pieces of medical literature and sixteen additional medical records. Pet'r's Exs. 72–90, ECF Nos. 53-2–53-3, 54-1–54-2, 56-1–56-14, 58-1; Pet'r's Ex. 92, ECF No. 58-3; Pet'r's Exs. 94–95, ECF Nos. 59-1–59-2. Respondent filed his second supplemental expert report by Dr. Donofrio on October 5, 2017.

2511769 (Fed. Cl. Spec. Mstr. May 24, 2019); *Sheets v. Sec'y of Health & Human Servs.*, No. 16-1173V, 2019 WL 2296212 (Fed. Cl. Spec. Mstr. April 30, 2019).

[4] Petitioner did not file this under an exhibit number; therefore, I will hereon refer to this as Pet'r's Second Expert Report.

Resp't's Ex. H, ECF No. 65. A week later, Petitioner filed four medical records. Pet'r's Exs. 98–101, ECF Nos. 66-1–66-4.

On November 13, 2017, I notified the parties by a written order that "no further hearings [would] be scheduled until further notice, and Chambers [would] reach out to the parties in early 2018 to schedule a hearing in this case." ECF No. 67. On December 19, 2017, Petitioner filed an additional medical record, an affidavit by Petitioner, an affidavit by Shelly Drobbin, and an affidavit by Kenneth Cohen, Ph.D. Pet'r's Exs. 102–105, ECF Nos. 68-1–68-4.

I issued a hearing order on November 9, 2018, setting the date for an entitlement hearing for April 17–18, 2019. ECF No. 72. On December 27, 2018, Petitioner submitted his pre-hearing brief. Pet'r's Pre-Hr'g Br. Two months later, Respondent filed his responsive pre-hearing brief. Resp't's Pre-Hr'g Br. ECF No. 79.

Petitioner filed an additional medical record on March 26, 2019. Pet'r's Ex. 106, ECF No. 80. Two days later, Respondent filed a piece of medical literature, an illustration for the hearing, and an updated CV for Dr. Donofrio. Resp't's Exs. J–L, ECF Nos. 81-1–81-3. On March 29, 2019, Petitioner filed his reply to Respondent's pre-hearing brief, a witness list, and a summary of previously filed articles. ECF Nos. 83–85. On April 1, 2019, Petitioner filed a medical report. Pet'r's Ex. 107, ECF No. 86. Between April 10 and April 12, 2019, Petitioner filed eight pieces of medical literature and a summary of previously filed articles. Pet'r's Exs. 108–114, ECF Nos. 88-1–88-7; Pet'r's Ex. 115, ECF No. 89-1.

A hearing was held on April 17–18, 2019. Transcript of Proc., ECF No. 92. Following the hearing, on April 22, 2019, Petitioner filed two medical records. Pet'r's Exs. 116–117, ECF No. 90. Respondent did not request any further post-hearing submissions. This matter is now ripe for consideration.

II. Factual Background

A. Petitioner's Statement

Petitioner did not testify during the hearing; however, he provided an affidavit. Pet'r's Ex. 103, ECF 68-2. Petitioner affirmed that he developed "a very high fever in the 102.5–103.0 range" on the same day that he received his flu vaccination. *Id.* at 1. Petitioner noted that he has received several flu vaccinations in the past, "none of which contained the H1N1 flu vaccination." *Id.* Petitioner affirmed that he took aspirin to address his fever and "after a period of time, [his] fever dissipated and returned to normal." *Id.*

Petitioner stated there was an incident at work within two weeks of his vaccination, wherein he "was unable to write." *Id.* at 2. Petitioner noted that his hands ultimately returned to normal. *Id.* Petitioner also described instances of muscle weakness in his arm during the ensuing months. *Id.* He noted that his "shoulders were getting weaker," and he decided to see his doctor, Zorica Mercadante, M.D., in January of 2012. *Id.* Petitioner listed multiple symptoms he developed "over the last six years since the Nov. 18, 2011 swine flu inoculation," including "fasciculations, a metamorphosis to [his] body which include[s] muscle atrophy in many muscle groups, and a significantly distended stomach." *Id.* Petitioner then described other symptoms that developed

over time, including back pain and "'crunchy' numb feelings in [his] toes, which [became] more painful and weak." *Id.* at 3. Petitioner described other complaints that were current as of the date of his affidavit, sworn to December 12, 2017. *Id.* at 4. He noted that he had suffered previously from "low back issues, chronic fatigue syndrome, seasonal allergies, and a number of prostatis (sic) and/or bronchial type infections" but affirmed that "these all were tested for and/or successfully treated and managed." *Id.*

B. Medical History

Petitioner's medical history prior to the vaccination is significant for episodes of shortness of breath, eosinophilic pleural effusion[5], chronic fatigue syndrome, chronic lower back pain, asthma, vertigo, muscle spasms, and L4-5-disc herniation. Pet'r's Ex. 5 at 3; Pet'r's Ex. 12 at 84–85. Petitioner's family history is significant for his son's carnitine palmitoyl transferase deficiency (CPT II)[6]. Pet'r's Ex. 34-2 at 49. Approximately one month prior to vaccination, on October 13, 2011, Petitioner was seen by Dr. Mercadante and assessed with muscle spasms, acute pharyngitis, and lumbago with sciatica.[7] Pet'r's Ex. 34-1 at 3. Petitioner was prescribed an antibiotic, nerve pain reliever, muscle relaxer, and beta blocker. *Id.*

On November 18, 2011, Dr. Mercadante administered an intramuscular flu vaccine in Petitioner's left deltoid. *Id.* at 1. Petitioner returned to Dr. Mercadante for general laboratory testing in December 2011. Pet'r's Ex. 34-4 at 138–40. There was no indication of vaccine-related pain. Petitioner continued laboratory testing on January 9, 2012, with no diagnostic abnormalities. Pet'r's Ex. 34-5 at 189.

Petitioner complained to Dr. Mercadante on January 26, 2012, of chest pain and muscle spasms, Pet'r's Ex. 34-2 at 51, and again on January 31, 2012, for "continuing rib pain at sides of chest wall cavity," *id*. at 49. Petitioner was tested for Lyme disease with abnormal results and was given treatment without a definitive diagnosis. Pet'r's Ex. 12 at 50–55. Petitioner tested negative or normal for creatine kinase, aldolase, ANA, c-reactive protein, Sjorgen's Ab, thyroid, B12, ACE, rheumatoid factor, hepatitis C, and erythrocyte sedimentation rate. *Id.* He was assessed with "Unspecified Arthropathy Involving Multiple Sites." Pet'r's Ex. 34-2 at 49.

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[5] Eosinophilic is defined as pertaining to a granular leukocyte. *Dorland's Illustrated Medical Dictionary* 629 (32nd ed. 2012) [hereinafter *Dorland's*]. Pleural effusion is defined as the escape of fluid from the blood vessels or lymphatics into the serous membrane enveloping the lungs and lining the walls of the pulmonary cavities. *Stedman's Medical Dictionary* 616 (28th ed. 2006) [hereinafter *Stedman's*].

[6] CPT II is defined as "an autosomal recessive disorder caused by mutations in the CPT2 gene. The late-onset or adult-onset form is the most common and is marked by rhabdomyolysis following prolonged exercise, fasting, or febrile illness." *Dorland's* at 297. Rhabdomyolysis is defined as "disintegration or dissolution of muscle, associated with excretion of myoglobin in the urine." *Id.* at 1637. Myoglobin is a "hemoprotein" that "combines with oxygen, stores it, and transports it to the mitochondria of muscle cells, where it generates energy by combustion of glucose to carbon dioxide and water." *Id.* at 1223.

[7] Lumbago is defined as "a nonmedical term for any pain in the lower back." *Dorland's* at 1076. Sciatica is defined as "a syndrome characterized by pain radiating from the back into the buttock and along the posterior or lateral aspect of the lower limb." *Id.* at 1678.

Petitioner had an MRI on February 15, 2012, that revealed degeneration and stenosis. Pet'r's Ex. 12 at 78–81. A second test for Lyme disease came back negative. *Id.* at 27–28.

On March 16, 2012, Petitioner was seen by neurologist Harold Weinberg with complaints of "achy, stiff feeling in his calves and forearms" and occasional "true cramps" for the past ten years. Pet'r's Ex. 23 at 1. Petitioner told Dr. Weinberg that he had begun "to have similar symptoms in his upper arms" since December of 2011. *Id.* at 2. Petitioner also described left hand numbness that resolved rapidly, large toe numbness, foot pain while walking, blurred vision, and eye tearing. *Id.* at 1–2. The examination record notes that Petitioner was on prescription medication for chronic fatigue syndrome. *Id.* at 1. Dr. Weinberg opined that "years of musculoskeletal discomfort now has developed more distal sensory symptoms in the legs and possible weakness in the arms"; he concluded that Petitioner's "examination is most consistent with a predominantly sensory neuropathy."[8] *Id.* at 3.

Petitioner's neurological exam from March 27, 2012, was "significant for length dependent sensory peripheral neuropathy and mild atrophy of the left calf when compared to the right." Pet'r's Ex. 3 at 2. Petitioner "show[ed] full strength and 2+ reflexes," but there was evidence of mild spinothalamic sensory loss in both feet." *Id.* at 3. Petitioner's EMG/NCS study was abnormal, "with electrophysiologic evidence of mild to moderate sensory axonal large fiber peripheral neuropathy affecting the right leg greater than the left arm." *Id.*

Petitioner was seen by Dr. Weimer, a colleague of Dr. Thomas Brannagan III, for follow-up on June 5, 2012. Pet'r's Ex. 29 at 12. Dr. Weimer discussed Petitioner's non-length dependent predominantly sensory neuropathy with him. *Id.* Petitioner discussed additional symptoms, specifically neck pain, muscle wasting, twitching, and hoarseness. *Id.* Dr. Weimer noted Petitioner's B6 levels and stated that if neuropathy is a result of B6 toxicity, "it may take months

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[8] A condensed reference guide on neuropathy is provided here to navigate the documentation of Petitioner's condition and diagnoses.

Neuropathy is a "functional disturbance or pathological change in the peripheral nervous system, sometimes limited to noninflammatory lesions as opposed to those of neuritis; the etiology may be known or unknown. Known etiologies include complications of other diseases (such as diabetes or porphyria), or of toxicity states (such as poisoning with arsenic, isoniazid, lead, or nitrofurantoin). The terms *mononeuropathy* and *polyneuropathy* may be used to denote whether one nerve or several are involved. A number of conditions may be called either neuropathies or polyneuropathies." *Dorland's* at 1268.

Small fiber neuropathy is a type of peripheral neuropathy that selectively affects the axons of small and poorly myelinated or small and unmyelinated (C) fibers. Pet'r's Ex 111 at 3.

Polyneuropathy is a "neuropathy of several peripheral nerves simultaneously." *Dorland's* at 1491.

Sensory neuropathy is defined as "neuropathy or polyneuropathy of sensory nerves." *Id.* at 1269. A sensory nerve is "a peripheral afferent nerve that conducts impulses from receptors on a sense organ to the termination of its axon in the spinal cord or brain." *Id.* at 1259.

Axonal is defined as "pertaining to an axon; most often used to describe the type of underlying nerve pathology responsible for generalized polyneuropathies. In this context, usually used incorrectly, i.e. "axonal polyneuropathy", rather than "axon loss polyneuropathy" (to distinguish the disorder from a "demyelinating polyneuropathy")." *Stedman's* at 191. Axon is the single process of a nerve cell that under normal conditions conduct nervous impulses away from the cell body and its remaining processes (dendrites). . . . In neurology and other clinical work, the term is also used as meaning dendrites, which term is seldom used clinically." *Id.*

Axonopathy is defined as "a disorder affecting primarily the axons of peripheral nerve fibers (although secondary demyelination occurs), in contrast to one affecting only myelin (myelinopathy)." *Id.*

or may be more time before the symptoms improve, after stopping the vitamin." *Id.* at 13. He also noted that "[n]o other underlying cause was found" and deferred further testing. *Id.*

In July of 2012, Petitioner was seen by several physicians at the Mayo Clinic and diagnosed with obstructive sleep apnea, shortness of breath, mild sensory or sensory predominant peripheral neuropathy, chronic fatigue syndrome, and muscle atrophy with no evidence of myopathy or neuromuscular disorder. Pet'r's Ex. 7 at 1. Petitioner was seen by various specialists for his symptoms and was examined by neurologist Kathleen McEvoy for his neuropathy.[9] *Id.* at 16. Dr. McEvoy noted that Petitioner has "seen two neurologists in New York who have found sensory neuropathy but have had no explanation for his motor symptoms." *Id.* Petitioner recounted his neck pain, wrist pain with writing, pinching pains in his forearms, and weakness that began in December of 2011. *Id.* He noted that prior EMGs and exams by NYU neurologists had revealed his sensory neuropathy "for which no identifiable cause was found." *Id.* Petitioner described a struggle with weight gain and a constant battle with fatigue. *Id.* Dr. McEvoy's impressions were that Petitioner's "EMG and exam are consistent with mild neuropathy for which there is no obvious cause on laboratory testing." *Id.* at 18. She explained to Petitioner that "a large proportion of neuropathies are cryptogenic." *Id.* Dr. McEvoy found "nothing to support a neurologic cause" of Petitioner's "muscle fatigue[,] lack of stamina[,] perceived weakness[,] and wasting of his muscles." *Id.* She "suspect[ed] that his symptoms of fatigue and discomfort may relate to central hypersensitization as seen in fibromyalgia" and noted, "[h]is history of chronic fatigue suggests a predisposition to such a disorder." *Id.* at 19.

Petitioner's EMG/NCS studies from July 2, 2012, found "minimal abnormalities . . . suggestive of the early manifestation of a generalized peripheral neuropathy." Pet'r's Ex. 10 at 6. Petitioner's B6 level was tested and measured 498 against a reference range of 20–125. *Id.* at 19–20. Dr. Brannagan concurred with Dr. Weinberg and found "electrophysiologic evidence for a mild predominantly sensory neuropathy, which is consistent with a . . . non-length-dependent pattern." *Id.* at 7. He also concluded that "[t]here is no electrophysiologic evidence for a focal mononeuropathy, myopathy, motor neuron disease[,] left cervical[,] or lumbosacral radiculopathy." *Id.*

Petitioner returned to Dr. Mercadante on July 12, 2012, with the results of his Mayo Clinic testing. Pet'r's Ex. 34-1 at 38. Dr. Mercadante noted that "they believe that the B6 is causal and can take 3–6 months." *Id.* During a July 26, 2012 visit, Dr. Mercadante noted that Petitioner may have the CPT II trait and added flua vaccine reaction with fever and bone pains to Petitioner's allergies in the medical record. Pet'r's Ex. 34-2 at 41,44.

On September 6, 2012, Petitioner saw Dr. Weimer to discuss Petitioner's possible status as a CPT II symptomatic carrier. Pet'r's Ex. 29 at 9. Dr. Weimer noted that Dr. Michio Hirano, Petitioner's son's treater, explained that he "had no experience with CPT II carriers with symptoms

[9] Dr. McEvoy's record contains handwritten references to Issues: 1. B6, 2. CPT-II, 3. Flu Shot, 4. Lyme, but it is unclear who wrote these additions and when. The writing continues on the next page of the record with various symptoms, some dates, and general notes.

other than exercise intolerance and related symptoms." *Id.* Dr. Weimer also noted that Petitioner's "sensory symptoms have receded with the reduction in B6." *Id.*

Dr. Mercadante examined Petitioner on October 18, 2012, after Petitioner reported that his trainer was concerned about "worse atrophy in left shoulder." Pet'r's Ex. 34-1 at 23. Petitioner also complained of more pain. *Id.* at 24. There was no finding of worsening atrophy, but Dr. Mercadante wrote "my gut feeling is that he has some underlying genetic myopathy that may have been worsened over this last year by the flu shot or some other viral illness or just from lack of sleep and stress." *Id.* at 24. Lab results revealed extremely high levels of CK[10] at 1449 and high levels of enzymes at 20.9 and myoglobin at 259. Pet'r's Ex. 12 at 9.

Petitioner was seen by Dr. Hiroshi Mitsumoto, Director of the ALS[11] Center at Columbia University on November 13, 2012. Pet'r's Ex. 6 at 1–2. Examination revealed atrophy in Petitioner's right ankle, parascapular muscles, and right and left calves. *Id.* at 2. Dr. Mitsumoto wrote that Petitioner "has very peculiar focal muscle atrophy, . . . and it is hard to find a clear cause." *Id.* Dr. Mitsumoto found "no evidence of multifocal motor neuropathy" but stated there is no way [to] test and confirm such a condition with current electrophysiological testing." *Id.* Despite Petitioner's complaints, Dr. Mitsumoto could not detect any muscle weakness. *Id.* He told Petitioner that "unfortunately, [he] could not come up with answer at this point." *Id.*

Dr. Weimer re-tested Petitioner's CK levels during his November 14, 2012 visit, with normal results. Pet'r's Ex. 29 at 5–7. Petitioner indicated that he had stopped exercise to keep his levels low. *Id.* at 5.

Petitioner continued to see various physicians to identify the cause of his symptoms and receive effective treatment.[12]

Date	**Physician Specialty**	**Diagnosis**	**Exhibit No.**
01/28/13	Weimer Neurology	Unclear etiology of subjective weakness and muscle cramping; additional testing for CPT II deficiency; mild peripheral neuropathy in lower extremities with no other obvious cause than B6 toxicity	29 at 3

[10] CK is the acronym for creatine kinase - an enzyme necessary for muscle control and concentration, particularly during the initiation of exercise. *Dorland's* at 429.

[11] ALS is the acronym for amyotrophic lateral sclerosis. *Dorland's* at 2108.

[12] Petitioner has a voluminous medical record that has been reviewed in its entirely; however, only relevant sections that help show the chronology and evolution of Petitioner's symptoms and diagnosis have been recited here. *See Moriarty ex rel. Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Human Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

03/13/13	Streit Internal Medicine	CFS, sensory neuropathy; muscle atrophy; Lyme disease	39 at 1
04/05/13	Bhatt Neurology & EMG	Length dependent sensory peripheral neuropathy; mild atrophy	3 at 2
05/02/13	Mercadante Internal Medicine	Partial expression of CPT II is cause of symptomology, but post 2011 decompensation may be due to flu vaccine	34-9 at 290
05/03/13	Mammen/Paik Neurology	Carpal median neuropathy and ulnar neuropathy; excessive amounts of B6 could contribute to some neuropathic symptoms; mild form of CPT II deficiency	5 at 7
07/16/13	Thomashow Neurologist	Idiopathic neuropathy; wondered if underlying muscle issue potentially triggered by flu vaccine	4 at 1
08/24/13	Cudkowicz Neurologist	There may be reports of vaccines accelerated perhaps motor or nerve problems with a fast mutation; Unlikely related to CPT II carrier, more likely autoimmune motor sensory neuropathy; A course of IVIg is reasonable.	11 at 2–3
06/24/14	Berini Neurologist	EMG evidence of mild changes consistent with a myopathy; Lyme disease possible contributor: Lyme disease can cause fatigue, myalgias, joint pains, and neuropathy, which can be associated with fasciculations. No neuropathy noted on evaluation	15 at 3
07/02/14	Hirano ALS Specialist	Mild chronic inflammatory polyneuropathy or residual symptoms of mild Guillain-Barré ("GBS") triggered by the vaccination; IVIg x 5 courses has improved the joint pain and myalgias suggesting an inflammatory process. Antibiotic treatment for presumed Lyme and Babesia infections	14 at 4
09/10/14	Bach Physical Medicine	Neuromuscular disease as a result of mitochondrial myopathy variant	21 at 2
09/18/14	Horowitz Internal Medicine	Progressive myopathy triggered by exposure to tick-borne illness and flu vaccine with autoimmune overlap	33 at 17

04/20/15	Jacobs Endocrinologist	No definitive diagnosis, constellation of motor and sensory symptoms	50 at 1–2
06/01/15	Zecca Allergist	Atrophy indicative of inflammatory process	48 R 3–4
07/17/15	Pavlakis Neuromuscular Medicine Fellow	Exercise intolerance, CPT II carrier, Multifocal motor neuropathy	52 at 5–6
08/19/15	Hirano ALS specialist	Mild chronic polyneuropathy or residual mild GBS triggered by flu vaccination; exercise intolerance, CPT II mutation (unlikely), mild myopathy	62 page 6
09/10/15	Pestronk Neurologist	Early length dependent larger fiber axonal polyneuropathy	54 at 6
10/20/15	Cheng Director of Headache and Neuropathic Pain Unit	Small fiber neuropathy with possible large fiber involvement	58 at 4
12/18/15	Corzo Physical Medicine and Rehabilitation	Chronic non-demyelinating small and large fiber polyneuropathy, plausible postsynaptic neuromuscular disease process of poorly documented etiology	66 at 5
02/23/16	Cudkowicz Neurologist	Evidence of small fiber neuropathy	68 at 2

III. Experts

A. Experts

a. Petitioner's Expert, Dr. Arthur Brawer[13]

Dr. Brawer received his medical degree from Boston University and completed his residency at Boston VA and City Hospital. Pet'r's Ex. 44 at 1, ECF 31-5. He completed a fellowship in arthritis and is board-certified in medical examination, internal medicine, and rheumatology. *Id.* Dr. Brawer is currently in private practice as the Director of the Rheumatology and Arthritis Clinic at Monmouth Medical Center in New Jersey. *Id.* He has previously served in academia as assistant clinical professor at Robert Wood Johnson Medical School in New Jersey

[13] Dr. Brawer did not testify at the hearing. Tr. 7:12–13. Respondent noted that Dr. Brawer's reports are part of the record and that Petitioner "is free to rely on those things." Tr. 21:23–22:1.

and Hahnemann/Drexel College of Medicine in Pennsylvania. *Id.* at 2. Dr. Brawer has also published several articles on rheumatoid arthritis and disease related to knee and breast implants. *Id.* at 2–4. Dr. Brawer submitted four expert reports in this case. *See* Pet'r's Exs. 40, 69, 91, 96.

b. Petitioner's Expert, Dr. Allan Rubenstein

Dr. Rubenstein received his medical degree from Tufts University and is board-certified in psychiatry and neurology. Rub. C.V.[14] at 2, ECF 48. Dr. Rubenstein's education includes residency at Columbia-Presbyterian Medical Center and post-graduate training on medical genetics and experimental mammalian genetics. *Id.* at 1. Dr. Rubenstein is currently a clinical professor of neurology and pediatrics at New York University Langone Medical Center, with over 40 years of experience as a practitioner. *Id.* at 4; Tr. 29:25, 30:2. As a professor, he "[e]stablished [the] first interdisciplinary clinic devoted to neurofibromatosis[15] in the world." Rub. C.V. at 3. He has also previously served as "a former examiner for the American Board of Neurology – Psychiatry" and has "written a number of publications, peer-reviewed . . . on a variety of types of autonomic neuropathies and neural tumor syndromes." Tr. 30:5–9. He has conducted research on the growth and development of neurofibromas. Rub. C.V. at 4. Dr. Rubenstein also has several patents related to the treatment of neurofibromatosis. *Id.* at 5. Dr. Rubenstein submitted three expert reports in this case and testified at the entitlement hearing. *See* Pet'r's Exs. 71, 93, 97; Tr. 29–108.

Dr. Rubenstein testified that "most of [his] career has been involved with [the] autonomic nervous system, peripheral neuropathy and peripheral nerve tumor syndromes." Tr. 30:2–4. Although Dr. Rubenstein stated that he has never treated a patient with post-vaccination neuropathy, he has "assessed a few patients, including [Petitioner] and another patient several years ago, who had a recurrent Guillain-Barré Syndrome following a vaccination." Tr. 31:8–12. Dr. Rubenstein testified that he has "seen over [his] career a substantial number of patients with autoimmune neuropathies." Tr. 30:13–14. Dr. Rubenstein was offered to testify as an expert in neurology, specifically "peripheral and autoimmune peripheral neuropathies as well as assessments of other patients who have had vaccine-related autoimmune peripheral neuropathies." Tr. 32:8–12.

Respondent objected to Dr. Rubenstein's purported expertise in vaccine-related autoimmune disease. Tr. 32:17–18. Dr. Rubenstein clarified that he had "seen or evaluated a handful of people who have had claims of post-vaccination polyneuropathy." Tr. 33:7–8. He testified that he is familiar with literature on vaccine-induced disorders but does not have direct knowledge of a link between vaccine and disease, either through research or clinical practice. Tr. 33:13–34:6.

c. Respondent's Expert, Dr. Peter Donofrio

[14] Petitioner filed Dr. Rubenstein's C.V. without an exhibit number.

[15] Neurofibromatosis is defined as a familial condition characterized by formation of a usually benign tumor of peripheral nerves. *Dorland's* at 1265.

Dr. Peter Donofrio received his medical degree from the Ohio State University. Resp't's Ex. B at 1. His postdoctoral training includes internal medicine and neurology residencies and a neuromuscular fellowship, and he holds board certifications in all three fields. *Id.* at 3. Dr. Donofrio is currently a professor of neurology at Vanderbilt University. Resp't's Ex. B at 1, Tr. 110:18–19. He is a member of the Medical Advisory Committee of the GBS/CIDP International Foundation, and Vanderbilt University Medical Center is a certified Center of Excellence for the GBS/CIDP International Foundation. Resp't's Ex. A at 1. Dr. Donofrio testified that he is the director of the Muscular Dystrophy Association Clinic, the ALD Clinic at Vanderbilt, and the EMG and Nerve Conduction Study Laboratory. Tr. 113:1–4. The majority of patients that Dr. Donofrio sees as a part of his clinical practice have peripheral neuropathy. Tr. 113:14–15. Dr. Donofrio has published articles, book chapters and a textbook on peripheral neuropathy.[16] Tr. 114:4–6. He has also "written two or three papers on vaccine-associated conditions, usually not as the first author, but several authors down the list." Tr. 114:15–17. Dr. Donofrio submitted three expert reports in this case and was admitted without objection to testify as an expert in neurology, electrodiagnostic medicine and neuromuscular medicine. Resp't's Exs. A, G–H; Tr. 115:1–8.

B. Expert Reports and Testimony

a. Dr. Brawer

Prior to providing a written expert report in this case, Dr. Brawer reviewed multiple medical records, "including, but not limited to neurology, immunology, allergy, internal medicine, pulmonology, radiology, physical therapy, electrophysiology, pathology, laboratory medicine, rheumatology, infectious disease, and otolaryngology." Pet'r's Ex. 40 at 1. Dr. Brawer also conducted a physical examination and obtained "an extensive history" from Petitioner. *Id.* In his first written report, Dr. Brawer wrote that "the evolution of muscle weakness and muscle atrophy has expanded the differential diagnosis to now include coexisting motor polyneuropathy overlapping with neuromuscular juncture (sic) dysfunction." *Id.* at 3.

Dr. Brawer continued in a supplemental report that Petitioner developed "features suggestive of a chronic inflammatory non-demyelinating polyneuropathy, the latter encompassing primary symptoms of muscle atrophy, weakness, pronounced fatigue, exercise intolerance, and fasciculations." Pet'r's Ex. 69 at 1. Dr. Brawer distinguished Petitioner's symptoms of fatigue from his muscle weakness and spasms. *Id.* at 2. He explained that fatigue is characterized by a desire to rest and not reduced strength inherent in muscle weakness. *Id.* Furthermore, spasms can occur with or without weakness or fatigue. *Id.* Dr. Brawer continued that "no less than a dozen examining physicians have documented in their medical records varying amounts of muscle atrophy developing subsequent to November 18, 2011." *Id.* at 3. Dr. Brawer stated that "at least one muscle biopsy . . . revealed denervation and reinnervation atrophy" and noted Petitioner "manifested spontaneous fasciculations in his left thigh muscle." *Id.* He summarized Petitioner's "most coherent diagnosis is now best represented by a chronic autoimmune inflammatory non-demyelinating polyneuropathy." *Id.* at 4.

[16] *See* Peter Donofrio, *The Textbook of Peripheral Neuropathy* (2012).

In his final supplemental report, Dr. Brawer opined that Petitioner's "small fiber neuropathy label is consistent with his sensory dysfunction, and the large fiber axonopathy is mostly (but not exclusively) consistent with his motor dysfunction." Pet'r's Ex. 96 at 4. He concluded, "the vaccination [Petitioner] received on November 18, 2011, is causally related to the autoimmune neuropathy." *Id.*

Dr. Brawer explained that autoimmune neuropathies can develop because "antigens of infectious agents can cross react with self-antigens present on a variety of body cells, including immunocompetent cells, thereby triggering systemic inflammatory reactions." Pet'r's Ex. 40 at 4. He identified this phenomenon as molecular mimicry and noted that this process "has been used to explain why numerous vaccines are capable of causing a whole host of varied autoimmune disease." *Id.* Dr. Brawer continued that "vaccines contain a sequence of amino acids sufficiently similar to self-antigens, thereby producing cross reactivity by the development of autoantibodies and activation of T cells." *Id.* In his report, Dr. Brawer identified several medical conditions that can be "triggered by a variety of bacterial and viral vaccine materials," including "rheumatoid arthritis, Guillain-Barré syndrome, multiple sclerosis, hemolytic anemia, thrombocytopenic purpura, and diabetes mellitus." *Id.* at 5. Dr. Brawer was careful to note that the types of controlled epidemiologic studies necessary to irrevocably prove vaccine-induced illness are impossible to devise in many instances, due to condition rarity and resources. He also noted that "the risks for neurologic and rheumatologic disorders caused by vaccinations . . . have not been refuted." *Id.*

Dr. Brawer identified three points as evidence of the "logical cause and effect showing that the vaccinations received by [Petitioner] on November 18, 2011, were the reason for his injuries." *Id.* First, Petitioner did not suffer from pre-existing systemic, neurologic, or neuromuscular conditions prior to vaccination. *Id.* Second, there is no other known entity that could be responsible for Petitioner's symptoms. *Id.* Third, Petitioner's neurologic symptoms began within an appropriate time post vaccination to support vaccine-induced injury. *Id.*

b. Dr. Rubenstein

In his reports, Dr. Rubenstein recounted Petitioner's genetic history and history of pre-vaccination hernias. *See* Pet'r's Exs. 71, 93, and 97. He then discussed Petitioner's fever the day of vaccination and later symptoms of weakness and muscle cramping in his legs. Pet'r's Ex. 93 at 1–4. Dr. Rubenstein noted that Petitioner "was treated for possible Lyme disease with oral antibiotics . . . with no improvement." *Id.* at 1. He identified an EMG performed on March 27, 2012, that showed "evidence of mild to moderate sensory axonal large fiber peripheral neuropathy affecting the right leg greater than left arm." *Id.* Dr. Rubenstein also noted Dr. Mitsumoto's note of "multifocal muscle atrophy, raising the possibility of a mononeuropathy multiplex syndrome." *Id.* at 2. He discussed Dr. Cudkowicz's diagnosis of an autoimmune process and subsequent monthly IVIg treatments and noted they resulted in "some improvement in his symptoms." *Id.*

Dr. Rubenstein wrote that Petitioner suffers from an autoimmune neuropathy that was causally related to his Fluzone vaccination. *Id.* at 3. He identified two reasons for his opinions: "1. There is a temporal relationship between the vaccination and the onset of his symptoms. 2. Autoimmune neuropathy is a rare but reported complication of vaccinations in general and

influenza vaccination specifically." *Id.* Dr. Rubenstein also relied on the opinions of two of Petitioner's treaters, Drs. Latov and Cudkowicz, who noted that the vaccine was a possible trigger, and Petitioner's neuropathy was autoimmune, respectively. *Id.* Dr. Rubenstein stated, "[w]hile the results obtained have varied, it is clear that [Petitioner] has had multiple studies, which document a mononeuropathy multiplex pattern of neuropathy, which has previously been reported to occur following influenza immunization, in addition to small fiber neuropathy." *Id.* at 3–4. An overlying left shoulder radiculopathy was also considered by Dr. Rubenstein, who stated that additional injury "further confuses [Petitioner's] clinical picture." *Id.* at 4.

Dr. Rubenstein testified that he agreed with Dr. Hirano's diagnosis that Petitioner suffers from "chronic axonal neuropathy, predominantly sensory non-length dependent type and predominantly small fiber." Tr. 36:1–3. Dr. Rubenstein noted that Petitioner "developed symptoms within two weeks" of vaccination and explained that this was consistent with post-vaccination autoimmune symptoms that can develop "[a]nywhere from a few days to four to six weeks" later. Tr. 41:21, 9. The medical record, according to Dr. Rubenstein, documents "abnormalities of [Petitioner's] motor and sensory fibers, [which are] part of the peripheral nervous system." Tr. 37:18–20. Dr. Rubenstein explained that "these were predominantly non-myelinated, small, fiber-sensory fibers with some motor components, which were not symmetric." Tr. 37:21–23.

Dr. Rubenstein asserted that Petitioner's "abnormalities, the clinical presentation, [and] the neurophysiological findings are all consistent with an attack of an autoimmune process induced by [the flu] vaccine on [Petitioner's] peripheral nerves causing him to have the various complaints that he has, some of which have responded to . . . immunoglobulin treatment." Tr. 38:1–6. Evidence of successful IVIg treatment supports the "thought that the process involves exposing a variety of antigens, which are shared between components of the vaccine with peripheral nerve components, either myelin or axonal, which the human body responds to by producing antibodies, which then attack those antigenic components." Tr. 39:3–8.

In his report, Dr. Rubenstein referred to "the same theory of immunologic cross-reactivity between antigen components of viruses and/or viral vaccines and peripheral nerve components, which has been postulated to explain the phenomenon of Guillain-Barré syndrome induced by various viruses." Pet'r's Ex. 71 at 2. He then made reference to filed literature "for a discussion" of "auto-immune mechanisms involved in virus-induced polyneuropathy" and molecular mimicry. *Id.* Dr. Rubenstein testified that this "process . . ., in which a variety of types of cellular and systemic responses of the body's cells respond to a variety of stimuli, which are considered to be foreign to the body . . . by producing . . . either cellular or humeral antibodies, which are in most cases meant to be protective, but in some cases, such as in the autoimmune polyneuropathies, are, in fact, disruptive." Tr. 40:2–12.

When asked about Petitioner's pre-existing chronic fatigue syndrome, Dr. Rubenstein stated that the condition is difficult to diagnosis and unclear in Petitioner's case. Tr. 67:6–10. Furthermore, even if Petitioner was correctly diagnosed with chronic fatigue syndrome, Dr. Rubenstein did not believe it was significant to Petitioner's neuropathy. Tr. 43:17–20. Dr. Rubenstein conceded that Petitioner's history of low back pain and unilateral foot weakness was

significant but noted that those symptoms were explained after an MRI revealed that Petitioner suffered from lumbosacral radiculopathy. Tr. 45:12–21. Petitioner's obstructive sleep apnea, high vitamin B6 levels, vitamin D deficiency, hypogonadism, and sarcopenia were all dismissed by Dr. Rubenstein as irrelevant to Petitioner's neuropathy. Tr. 47:1–49: 9. Dr. Rubenstein identified Petitioner's "muscle cramps, intermittent weakness, numbness in his legs, and inability to function at a preexisting level" as "complaints that . . . relate" to Petitioner's post-vaccination condition. Tr. 60:1–4. Dr. Rubenstein also discussed symptoms that Petitioner reported developed within two weeks of vaccination. When asked about Petitioner's report of post-vaccination high fever followed by weakness, muscle cramping, and exercise intolerance, Dr. Rubenstein stated that to his knowledge, none of Petitioner's medical records generated in the year post vaccination contain any reference to these symptoms or the flu vaccine. Tr. 68:14–23. Dr. Rubenstein clarified that Petitioner reported these symptoms to him during an examination on August 9, 2016. *See* Tr. 71–72.

During cross-examination, Dr. Rubenstein testified that he "was the first one to state that the specific – specifically that this was a mononeuropathy multiplex presentation." Tr. 75:24–76:2. While he did not provide a definition through direct testimony, Dr. Rubenstein agreed that mononeuritis multiplex includes a group of disorders with asymmetrical sensory and motor peripheral neuropathy involving damage to at least two separate nerve areas. Tr. 76:8–12. He agreed that mononeuropathy multiplex is associated with viral hepatitis infections and Lyme disease. Tr. 76:17–21. When asked about Petitioner's course of treatment for Lyme disease, Dr. Rubenstein noted that it did not improve Petitioner's symptoms. Tr. 77:1–2. Dr. Rubenstein then explained that he "would not technically consider small fiber neuropathy of the type that [Petitioner] was documented as having as part of a mononeuropathy multiplex clinical presentation." Tr. 77:11–15. Petitioner's condition was not considered by Dr. Rubenstein to be demyelinating, but he did not rule out categorizing Petitioner's neuropathy as inflammatory. Tr. 79:16–19.

Dr. Rubenstein clarified during my questioning that there is evidence to support diagnoses of small fiber neuropathy and mononeuropathy multiplex. *See* Tr. 93. He further explained that both conditions occurred subsequent to Petitioner's vaccination but not necessarily simultaneously. Tr. 94:16–17. While unable to identify the onset for either condition, Dr. Rubenstein ultimately opined "that all the symptoms that Petitioner complained about within the first month subsequent to immunization, [are] consistent with the presentation of either small fiber neuropathy or mononeuropathy multiplex." Tr. 95:1–5. Dr. Rubenstein stated that he "can't tell which of the various problems subsequently documented were represented by the complaints which were made in the first four weeks." Tr. 96:7–10. However, "at various times over at least a five year — no . . . eight-year period, [Petitioner] has been identified as having, by various diagnostic tests, all of those issues." Tr. 95:11–14. Dr. Rubenstein testified that ultimately, Petitioner "was electrophysiologically diagnosed with having both those entities." Tr. 95:6–7.

Petitioner filed one article that discusses mononeuritis multiplex. Pet'r's Ex. 92 at 20–22.[17] The article describes a case study of a 72-year-old woman who presented with a "history of progressive distal arm and leg symptoms, tiredness, and anorexia." Pet'r's Ex. 92 at 20. One-week post vaccination, she "experienced pain in her left buttock which radiated down her left leg." *Id.* She later developed weakness in both hands and her left foot became numb and weak. *Id.* Her presentation was "indistinguishable . . . from axonal Guillain-Barré syndrome." *Id.* "Nerve conduction studies carried out on admission confirmed a peripheral neuropathy with features of a mononeuritis multiplex syndrome." *Id.* Ultimately the patient had "severe distal weakness" and "was very disabled, unable to walk or use either hand." *Id.*

In his report and testimony, Dr. Rubenstein drew analogies between flu vaccine-induced GBS and Petitioner's condition. When asked whether there was a relationship between the wild flu virus and Petitioner's condition like the causal relationship between influenza and GBS, Dr. Rubenstein stated that he was "unaware of any published evidence for either of [Petitioner's conditions] to [his] knowledge, being reported as a result of influenza per se." Tr. 100:20–23. When asked why there did not appear to be such a relationship with respect to the virus, Dr. Rubenstein stated simply, "I don't have an explanation for it." Tr. 101:2. Dr. Rubenstein testified that "it's certainly plausible that there are associations which have been identified or reported[,] but that's purely speculation on my part." Tr. 101:5–8. The analogy between GBS and Petitioner's condition works, according to Dr. Rubenstein, because of the pathogenic mechanism seen in both of these autoimmune neuropathic conditions. Dr. Rubenstein testified that his reason for identifying Petitioner's neuropathy as autoimmune was due to the temporal relationship between vaccine and clinical symptomology, other examples of peripheral nerve phenomenon following the flu vaccine, the opinion from several of Petitioner's treaters that he had an autoimmune neuropathy, and the improvement of Petitioner's symptoms following treatment commonly used for autoimmune neuropathies. Tr. 102:7–20.

c. Dr. Donofrio

In his initial expert report, Dr. Donofrio commented that Petitioner had several physicians including specialists in neuromuscular disorders, internal medicine, pulmonary disease, infectious disease, and rheumatology, and no consensus was reached, nor could anyone "secure a diagnosis that explains the Petitioner's symptoms." Resp't's Ex. A at 4. Dr. Donofrio acknowledged that several of Petitioner's treating neurologists "found a mild sensory peripheral neuropathy on examination and by nerve conduction studies and EMG." *Id.* However, he asserted that Petitioner's complaints of "muscle atrophy, weakness, pronounced fatigue, exercise intolerance and fasciculations" are not explained by sensory peripheral neuropathy. *Id.* Dr. Donofrio also noted that "[s]everal of the neuromuscular experts have openly stated that they could not explain the [Petitioner's] symptoms by a nerve, muscle, or neuromuscular disorder." *Id.* Petitioner's medical history was discussed in detail, and Dr. Donofrio stated that an EMG study conducted on

[17] J.H.K. Hull et al., *Severe Vasculitic Neuropathy Following Influenza Vaccination*, 75 J. Neurol. Neurosurg. & Psychiatry 1507 (2004).

August 26, 2015, showed no evidence of generalized polyneuropathy, radiculopathy, or myopathy. *Id.* at 5. He also discussed the lack of evidence for diffuse neuropathy at that time. *Id.*

Dr. Donofrio noted that the 2012 Institute of Medicine textbook on Adverse Effects of Vaccines stated, "the epidemiologic evidence is insufficient or absent to assess an association between influence vaccination and small fiber neuropathy." *Id.* at 6. In his report, Dr. Donofrio concluded that Petitioner's weakness and loss of muscle mass were caused by vitamin D deficiency and low testosterone, coupled with his pre-existing conditions, including chronic fatigue syndrome. *Id.* at 7. Furthermore, Dr. Donofrio dismissed Petitioner's theory of molecular mimicry for two reasons: 1) without a definite diagnosis, it would be impossible to hypothesize an antigen to relate to antibodies and 2) no neurologist has found evidence of an autoimmune process in Petitioner's case. *Id.*

After reviewing updated medical records, Dr. Donofrio wrote in a supplemental report, "[t]his reviewer agrees that the [P]etitioner had pathologic evidence for a small fiber neuropathy but did not have typical symptoms." Resp't's Ex. H at 7. However, "[c]onsidering the delay," Dr. Donofrio concluded, "the findings on skin biopsy cannot be linked to the flu vaccination with confidence as other conditions may have arisen to produce the skin biopsy changes." *Id.* He also asserted that "a positive response of the Petitioner to IVIG does not support an autoimmune neurologic explanation for the [P]etitioner's symptoms." *Id.* at 8. He noted that "improvement might be explained by a rheumatologic process," and a peripheral neuropathy would not explain all of Petitioner's symptoms. *Id.*

Dr. Donofrio's testimony began with a discussion of Petitioner's medical records immediately before and after vaccination. Dr. Donofrio noted that Petitioner saw his primary care physician, Dr. Mercadante, approximately one-month pre vaccination and was assessed with "spasm of muscle, acute pharyngitis, lumbago . . ., sciatica, and then a current assessment of allergic rhinitis, cause unspecified." Tr. 119:5–8. Dr. Donofrio noted that the vaccination record was uneventful and turned to Petitioner's January 2012 visit. Tr. 120:9–11. Petitioner was diagnosed with chest pain and gastroesophageal reflux disease, but "there was really no comment about neurologic symptoms or signs." Tr. 120:21–22. Dr. Donofrio acknowledged the March 1st referral to a neurologist but added that he did not see "an examination." Tr. 121:24. He noted that the referral included "some symptoms in December, holding [Petitioner's] grandchild [and] some numbness in the left toe." Tr. 122:20–21.

The first indication of any neuropathy appeared following testing after a March 16, 2012 visit for extremity discomfort. *See* Tr. 122–123. The data from the testing shows "abnormalities in sensory nerves[, i]n fact, every sensory nerve." Tr. 123:20–21. Dr. Donofrio did not feel this was relevant to Petitioner's symptoms, because "there is no comment in the note by Dr. Weinberg when [the symptoms] began. And [he] didn't see any temporal relationship between this and the vaccination that was done." Tr. 124:14–17. When asked if these symptoms could have predated Petitioner's vaccination, Dr. Donofrio stated, "[e]asily." Tr. 124:18–19. Dr. Donofrio added that "none of the symptoms of the [P]etitioner are in line with the sensory neuropathy that was found on exam and corroborated by the nerve conduction studies." Tr. 125:1–4. He was "not sure what [Dr. Weimer] meant by" his mild sensory neuropathy conclusion during an examination six-

months post vaccination. Tr. 127:24–25. However, Dr. Donofrio opined "that B6 toxicity can cause a sensory neuropathy." Tr. 128:16.

Dr. Donofrio continued to discuss Petitioner's extensive medical records and pointed out a lack of consensus or clear diagnosis among treaters. Dr. McEvoy, a Mayo Clinic neurologist, found "a distal loss of sensation consistent with a length-dependent sensory neuropathy" on July 3, 2012. Tr. 133:9–11. However, "she says there is nothing to support a neurologic cause of [Petitioner's muscle fatigue, lack of stamina, weakness, and wasting of muscles]." Tr. 134:14–17. Dr. Donofrio noted that the first reference to Petitioner's vaccination he found appeared in a July 26, 2012 record from Dr. Mercadante. Tr. 136:20. Notwithstanding that note, Dr. Mercadante's assessment of Petitioner's condition is "unspecified idiopathic peripheral neuropathy." Tr. 137:2. Additionally, Dr. Donofrio noted Dr. Litchy's opinion that Petitioner's muscles of concern "were mostly atrophic," Tr. 138:11–2, and that Dr. Mitsumoto "could find no evidence of weakness and could not find any atrophy that was impressive to him[;] he didn't have any explanation for the patient's symptoms[,]" Tr. 140:19–21. Dr. Donofrio stated that in a medical note from an examination approximately one-year post vaccination on November 13, 2012, he "[could not] find any neurological disease that explains the predominant symptoms that the [P]etitioner has." Tr. 141:2–3. Reviewing Petitioner's symptoms years after the vaccination, Dr. Donofrio testified that testing done by the Mayo Clinic in 2014 yielded "very normal amplitudes." Tr. 143:14–15. Dr. Donofrio continued that a repeat study in 2015, almost four years post vaccination, "didn't show any abnormalities in terms of nerve conduction study results." Tr. 143:17–19.

Dr. Donofrio could not explain Petitioner's response to IVIg and stated, "I would wonder if [Petitioner] didn't have a rheumatological disease." Tr. 144:24–25. None of Petitioner's doctors could identify a diagnosis that explained all of Petitioner's symptoms, and neither could Dr. Donofrio. Tr. 146:4.

Dr. Donofrio confirmed that Petitioner "has a small fiber neuropathy, because you have a skin biopsy here that showed a reduction in what we call the epidural sensory density." Tr. 149:20–22. He went further and stated that "based on some of the nerve conduction studies, we can say there's a small fiber and a large fiber sensory neuropathy." Tr. 150:3–5. However, Dr. Donofrio asserted that "not a single nerve conduction study showed abnormalities in the motor fibers of any significance." Tr. 150:11–13. Petitioner's major symptoms were weakness and fatigue, not devastating nerve and limb pain with inflammation. *See* Tr. 155–156. Dr. Rubenstein defined Petitioner's condition as small fiber neuropathy plus an autoimmune mononeuritis multiplex; however, Dr. Donofrio explained that "the picture doesn't fit well to explain this man's symptoms." Tr. 156:7–8. Dr. Donofrio defined the elements of mononeuritis multiplex: "so mononeuropathy [refers to] a single nerve that is affected . . . median nerve, ulnar nerve, radial nerve, et cetera[;] mononeuritis multiplex means . . . two or more[; a]nd then mononeuritis implies inflammation." Tr. 155:24–25. He continued that this condition is "common in autoimmune disorders and in rheumatological disorders," Tr. 156:3–4, but they "are devastating [and] commonly associated with a lot of nerve pain and limb pain[,]" Tr. 156:6–7.

Petitioner's muscle weakness was countered, in Dr. Donofrio's opinion, with "the physical examination [that] stated no atrophy and normal strength in every muscle detected." Tr. 157:12–

13. Dr. Donofrio added that Petitioner had two normal MRIs making it "hard for [Dr. Donofrio] to explain [Petitioner's] weakness when the most recent neurologist who saw him didn't find him to be weak at all and didn't find any reason for atrophy." Tr. 159:19–22.

Dr. Donofrio did not believe that Petitioner's timeline was consistent with the development of a neuropathy caused by vaccine-induced molecular mimicry because "if [Petitioner] had a reaction of any significance, . . . he would have seen his primary care doctor in November or December. Or when he went to the hospital . . ., he would have mentioned not only the reaction but feeling weak and having atrophy." Tr. 160:14–19. Dr. Donofrio testified that "it's pretty hard for [him] to accept a reaction to the vaccination that involves any part of the nervous system that would not have caused [Petitioner] to see a doctor for at least three and a half to four months." Tr. 161:3–6. He stated that he would anticipate symptoms indicative of an autoimmune response to appear "[a]bout seven to ten days after the vaccination." Tr. 182:22. He was critical of Dr. Mercadante's failure to note Petitioner's symptoms in November through March of 2012 and presumed "[P]etitioner wasn't having the symptoms or didn't tell the physician or they weren't documented for some reason." Tr. 164:6–8.

Dr. Donofrio did not believe it was possible to determine when Petitioner's condition developed because small fiber neuropathy can be asymptomatic, and Petitioner's skin biopsy confirming his condition was completed four years post vaccination. Tr. 180:13–14, 174:3–6.

When asked about Dr. Hirano's diagnosis, Dr. Donofrio testified that he did not agree that Petitioner's "most parsimonious diagnosis is a chronic axonal neuropathy" that was "supported by improvement of joint pain and myalgias." Tr. 196:7–9. However, he did concede that the flu vaccine can cause an axonal form of peripheral neuropathy in "a very rare presentation." Tr. 166:5. Dr. Donofrio stated that "it would either have to be an antibody or what we call a T cell reaction against the epitopes and antigens for molecular mimicry to induce neuropathy, such as with GBS." Tr. 171:21–23. He further explained that "to prove that [with respect to axonal peripheral neuropathy], you have to identify and measure the antibody that causes the disease in the individual patient who has the problem." Tr. 172:88–11. After setting out this test to prove causation, Dr. Donofrio conceded that the testing currently being conducted with respect to axonal peripheral neuropathy and the relevant epitopes and antibodies is not adequate. Tr. 172:12–15.

Dr. Donofrio agreed that if Petitioner did experience the onset of his symptoms in December of 2011, the timing, although not determinative, would be appropriate for vaccine induction. Tr. 183:16–20. Despite the temporal relationship, Dr. Donofrio did not believe Petitioner's actual symptoms to be consistent with sensory axonal peripheral neuropathy and testified that he "would struggle to relate [Petitioner's symptoms] to the vaccine." Tr. 183:25. He explained that, if that was the case, Petitioner should have developed "numbness, tingling, and pain in the soles of his feet and toes and not proximal weakness in the arms trying to hold his grandchild." Tr. 184:1–5. Dr. Donofrio testified that he would "expect to see in an autoimmune neuropathy an inflammatory reaction between the antibodies and the antigen." Tr. 203:7–9. He agreed that fever is indicative of inflammation and stated that if Petitioner's symptoms were indicative of an autoimmune neuropathy, he "would expect to see a fever on the night of the vaccination." Tr. 204:11–16. Dr. Donofrio did not discount Petitioner's assertion that he did

experience a fever the night of vaccination but noted that Petitioner did not relay this symptom to his doctors. "And why would someone who felt that they had a reaction to the vaccine who was having active symptoms not mention them to a healthcare provider?" Tr. 12-15. Dr. Donofrio also did not think that motor peripheral neuropathy was a good fit, because he would expect Petitioner's legs to be affected, except in the unusual case. Tr. 184:7–17. He accepted Petitioner's account that he developed "crunchy, numb feelings in [his] toes[,] which [became] more painful and weak" but found it odd that Petitioner did not report this to his doctor. Tr. 187: 9–13.

Petitioner's small fiber neuropathy was confirmed by biopsy, but Dr. Donofrio did not support the conclusion that Petitioner also had mononeuritis multiplex, "because the constellation of symptoms is usually rapid onset, horrible pain, lots of numbness and tingling in the distribution of a single nerve." Tr. 202:8–11. Dr. Donofrio testified that he did not think Petitioner's symptoms fit. Tr. 202:12.

V. Applicable Legal Standard

To receive compensation under the Vaccine Act, Petitioner must demonstrate either that: (1) his condition is a "Table Injury," and therefore resulted from the receipt of a covered vaccine or vaccines within the time frame prescribed by the Vaccine Injury Table set forth at § 14, as amended by 42 C.F.R. § 100.3; or (2) Petitioner's condition is an "off-Table Injury," one not listed on the Table, that resulted from his receipt of a covered vaccine. *See* § 11(c)(1)(C); *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1319–20 (Fed. Cir. 2006). Petitioner's claim that his flu vaccination caused his symptoms does not fall within the Vaccine Table. Thus, it must be proven that his vaccine was the cause-in-fact of his condition.

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that his vaccine was the cause of his injury. § 13(a)(1)(A). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was a "substantial factor" and a "but for" cause of the injury is enough for recovery. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999).[18]

In cases where the diagnosis is contested, "special masters may find whether a preponderance of evidence supports any proposed diagnosis before evaluating whether a vaccine caused that illness." *Hibbard v. Sec'y of Health & Human Servs*, No. 07–446V, 2011 WL 1766033, at *6 (Fed. Cl. Spec. Mstr. April 12, 2011) (citing *Broekelschen v. Sec'y of Health & Human Servs.,* 618 F.3d 1339, 1345–46 (Fed.Cir.2010)).

In *Althen v. Sec'y of Health & Human Servs.*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d 1274, 1278 (Fed. Cir. 2005). The *Althen* test requires a petitioner to set forth: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical

[18] The Vaccine Act also requires petitioners to show by preponderant evidence that the "residual effects or complications" of the alleged vaccine-related injury lasted for more than six months. § 11(c)(1)(D)(i). It is undisputed that this six-month requirement is satisfied in this case.

sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *See id.* (internal citations omitted).

Specifically, under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question "can [the] vaccine(s) at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *aff'd*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006), *cert. denied*, 551 U.S. 1102 (2007). This may be accomplished in a number of ways. "Reliability and plausibility of . . . pathogenesis can be bolstered by providing evidence that at least a sufficient minority in the medical community has accepted the theory, so as to render it credible." *Id.* Additionally, "epidemiological studies and an expert's experience, while not dispositive, lend significant credence to the claim of plausibility." *Id.* Medical literature published in respected medical journals is also persuasive. *Id.* "However, publication 'does *not* necessarily correlate with reliability', because 'in some instances well-grounded but innovative theories will not have been published.'" *Id.* (quoting *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 593–94 (1993) (emphasis in original)).

In addition to showing that the vaccine at issue can cause a particular injury, a petitioner must also, under *Althen's* second prong, prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1278. A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original) (internal citations omitted).

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). This is because "treating physicians are likely to be in the best position to determine whether 'a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events." *Cucuras v. Sec'y of Health and Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). While a special master must consider these opinions and records, they are not "binding on the special master or court." 42 U.S.C. § 300aa-13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record . . . ." *Id.*

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Human Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992) ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period. . . . Without

more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

A petitioner who satisfies all three prongs of the *Althen* test has established a prima facie showing of causation. *Hammitt v. Sec'y of Health & Human Servs*., 98 Fed. Cl. 719, 726 (2011). Consequently, the burden then shifts to the government to prove that an alternative cause, unrelated to the administration of the vaccine, was the "sole substantial factor" in causing the alleged injury. *De Bazan*, 539 F.3d at 1354; *see also Hammitt*, 98 Fed. Cl. at 726 (explaining that Respondent's burden is to show that the "factor unrelated" was the "sole substantial factor" in causing the injury). Additionally, a factor unrelated "may not include 'any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness or condition.'" 42 U.S.C. § 300aa-13(a)(2); *see also Doe v. Sec'y of Health & Human Servs.*, 601 F.3d 1349 (Fed. Cir. 2010) (opining that an idiopathic diagnosis cannot be a "factor unrelated," as it is idiopathic).

VI. Discussion

A. Diagnosis

Prior to evaluating the probative value of evidence to explain the how and why of Petitioner's injuries, an analysis of the relevant proposed injuries is necessary. Petitioner was diagnosed initially with sensory neuropathy by Dr. Weinberg. This diagnosis was also made by several other physicians, including neurologists Weimer and Paik, and Petitioner's small fiber neuropathy was ultimately confirmed by biopsy. The opinions of these treaters and others, Petitioner's biopsy testing, and the expert consensus of Drs. Rubenstein and Donofrio provide preponderant evidence that Petitioner suffers from small fiber neuropathy, which is a type of peripheral sensory neuropathy. While Petitioner's treaters and the experts largely agree that he suffered from neuropathy, they disagree on further specifics of his diagnosis, including symptom onset, symptom etiology, and comorbidity.

Petitioner alleged that as a result of his vaccination, he also suffers from neuromuscular junction dysfunction. The evidence and testimony that he presented did not adequately discuss the development of this condition or identify relevant symptoms that Petitioner suffered from. Undoubtably, Dr. Brawer relied on several notations in the record that Petitioner suffered from some form of neuromuscular disease. However, there was no attempt by Dr. Brawer to explain how he came to this specific diagnosis, its relationship to Petitioner's small fiber neuropathy, or how the asserted causation theory applies. Dr. Rubenstein then attempted to tie all the symptoms Petitioner reported over the ensuing years into one, all-inclusive neuropathic complex, to include sensory and motor polyneuropathy, neuromuscular junction dysfunction, small fiber neuropathy, autoimmune peripheral neuropathy, large fiber neuropathy, and mononeuritis multiplex. This conglomeration attempts to account for all of Petitioner's symptoms, but instead, obscures the symptom onset for Petitioner's confirmed diagnosis of small fiber neuropathy. As described, Petitioner's assertion of various neuromuscular conditions is not supported by medical records that correlate specific symptoms with diagnoses. Furthermore, the etiology for a vaccine-induced neuromuscular junction dysfunction, for example, is underdeveloped. Ultimately, Petitioner has

not provided preponderant evidence that he suffers from any of the other conditions asserted in his petition.

Finally, although not alleged in the petition, Petitioner presented expert opinion and testimony that he suffers from mononeuritis multiplex. I attempted to clarify Dr. Rubenstein's diagnosis, but he only broadened the general definition upon further questioning. Dr. Rubenstein vaguely defined mononeuritis multiplex as "a clinical syndrome," Tr. 93:4, that "refers to multiple individual nerves being affected[,]" Tr. 93:12–13. He agreed that it was an umbrella term with symptoms found in several other conditions. Tr. 93:4–7. Dr. Rubenstein did not say which of Petitioner's individual nerves were affected and could only state there was "evidence for multiple other nerves being affected which may be part of this and likely are." Tr. 93:23–24. He also could not point to specific symptoms that relate the onset of Petitioner's mononeuritis multiplex to the vaccine and clarified, "I can't tell which of the various problems subsequently documented were represented by the complaints which were made in the first four weeks." Tr. 96:7–10. Dr. Rubenstein did not explain the basis for his diagnosis, nor did he provide a logical sequence of cause and effect showing that the vaccination was the reason for Petitioner's mononeuritis multiplex. There is one article filed that describes a case of mononeuritis multiplex following vaccination. The case study is not analogous to Petitioner's, but it is consistent with the symptomology and presentation that Respondent's expert, Dr. Donofrio, described. It does not provide support that Petitioner suffered from this diagnosis, nor does Petitioner provide preponderant evidence that his vaccination caused him to develop this disorder.

B. *Althen* Prong 1

Petitioner's theory of causation was first introduced by his expert, Dr. Brawer. Although Dr. Brawer did not testify, he provided expert reports, which Respondent responded to. Furthermore, Respondent did not object to the consideration of Dr. Brawer's reports despite his failure to testify. Dr. Brawer's opinion that autoimmune neuropathies can develop as a result of vaccination has been supported in the Program by other entitlement decisions. *See Salmins v. Sec'y of Health & Human Servs*., No 11-140V, 2014 WL 1569478 (Fed. Cl. Spec. Mstr. Mar. 31, 2014); *Jane Doe/06 v. Sec'y of Health & Human Servs.*, No [redacted]V, 2007 WL 3120297 (Fed. Cl. Spec. Mstr. Oct. 18, 2007); *Land v. Sec'y of Health & Human Servs.,* No 12-474V, 2014 WL 2488705 (Fed. Cl. Spec. Mstr. May 13, 2014). Petitioner also filed articles to further explain the concept of molecular mimicry as it could apply to the development of small fiber neuropathy. Those articles were not discussed in any detail during the hearing, nor were they discussed in any of the expert reports. However, both Petitioner's and Respondent's experts attested to the viability of molecular mimicry as a cause for the development of certain neuropathies. Dr. Brawer wrote that there is medical literature published over the last several decades "[l]ending evidence to the cause and effect theory that vaccinations [influenza, tetanus toxoid, diphtheria, etc.] produce injury [rheumatoid arthritis, systemic lupus erythematosus, reactive arthritis, Guillain-Barré Syndrome, multiple sclerosis, etc.] via a molecular mimicry mechanism." Pet'r's Ex. 40 at 5. Dr. Rubenstein testified that "[m]olecular mimicry is a pathogenic process proposed for autoimmune diseases of a variety of types, and peripheral neuropathies specifically, which is basically applied to the

process we just discussed." Tr. 41:1–4. Additionally, Dr. Donofrio was asked if the flu vaccine can cause an axonal peripheral neuropathy that presents as GBS. He stated, "[i]t is one of the many forms of peripheral neuropathy as long as the patient's presentation fits Guillain-Barré." Tr. 165:5–7. All three experts are esteemed members of the medical community. Petitioner has established by a preponderance of the evidence that the flu vaccine can cause the development of some peripheral neuropathies via molecular mimicry.

Establishing that molecular mimicry is a logical pathogenesis for an axonal peripheral neuropathy that is diagnosed as GBS is, however, only the first step for Petitioner. Petitioner must also establish that this theory of vaccine-induced neuropathy is relevant to the type of neuropathy diagnosed in this case. Both of Petitioner's experts maintain that Petitioner's small fiber neuropathy is a peripheral neuropathy that can be autoimmune in nature. Indeed, the nature of pathogenic molecular mimicry would suggest that some sort of immune response develops into autoimmune disease. Dr. Donofrio's concession regarding the relationship between molecular mimicry and vaccine-induced injury was limited, and he did not subscribe to the application of molecular mimicry to neuropathies in general. He did not state that he specifically believed the flu vaccine could lead to the development of autoimmune small fiber neuropathy; he was "not sure [he] could say that" the flu vaccine can cause the axonal form of peripheral neuropathy similar what Petitioner has. Tr. 165:19–25.

Dr. Donofrio's hesitation appears to be based largely on the rarity of vaccine-induced injury. He noted that the only way to prove to his satisfaction that molecular mimicry occurs with respect to any condition would be to identify the specific antibodies and epitopes that are cross-reacting. Tr. 171:21–23. This level of specificity is beyond what is required to meet the preponderant standard in the Program. For special masters "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program." *Knudsen*, 35 F.3d at 549. A Petitioner is tasked, however, with presenting a "sequence of cause and effect [that] is logical and legally probable[.]" *Id.* at 548–49 (internal citations omitted). In other words, to the extent that a Petitioner does present a specific biological mechanism, it must be logical. It is logical that GBS is an appropriate analogy to other forms of autoimmune axonal, peripheral neuropathies. It is also logical to draw comparisons between the eitology of diseases that are so similar that doctors sometimes misdiagnose one for the other. Indeed, Dr. Donofrio agreed that these conditions don't always follow the classic pattern of presentation. Tr. 184:15–17. This makes it all the more difficult to determine etiology based on the progression of a specific case or to accept the contention that an unknown etiology is an acceptable alternative cause.

Dr. Donofrio's assertion that molecular mimicry can only be considered as a viable causation theory when the cross-reaction has been identified and tested to prove pathogenesis is too high a standard. Petitioner's experts have presented a theory of molecular mimicry. Petitioner has presented evidence to show that the medical community has determined molecular mimicry can cause some flu-vaccine-induced peripheral neuropathies. Petitioner has drawn parallels between atypical axonal GBS and autoimmune small fiber neuropathy to illustrate how the pathogenesis of one can instruct with respect to the other. Respondent has not offered a specific

critique of the use of molecular mimicry to explain vaccine induced small fiber neuropathy. Respondent does not identify any contradictions in Petitioner's theory. Petitioner has met his burden with preponderant evidence that molecular mimicry resulting from the flu vaccine can cause the type small fiber neuropathy Petitioner suffers from.

Dr. Rubenstein went further and argued that Petitioner's mononeuritis multiplex is a comorbidity that was also vaccine-induced via molecular mimicry. There was no real discussion on how molecular mimicry has been specifically linked to mononeuritis multiplex. Dr. Brawer did not provide an opinion, and Dr. Donofrio did not find a discussion of that condition to be of any relevance to Petitioner. Dr. Rubenstein's discussion of this condition appeared to be an attempt to explain Petitioner's atypical symptoms. It was not clear how Dr. Rubenstein was defining mononeuritis multiplex as distinct from a peripheral small fiber neuropathy or how it is similar or different from the GBS analogy. I find that Petitioner did not establish, by a preponderant standard, a causation theory as it relates to mononeuritis multiplex.

C. *Althen* Prong 2

As an initial matter, Dr. Brawer's three-prong rationale for concluding Petitioner's neuropathy was vaccine-induced is incomplete, because it does not adequately contemplate the record. Dr. Brawer wrote that Petitioner did not suffer from any pre-existing systemic neurologic or neuromuscular conditions; however, Dr. Brawer did not discuss Petitioner's assertion that he had a ten-year history of achy, stiff feelings in his calves and forearms with occasional true cramps. In fact, Petitioner characterized post-vaccination symptoms in his upper arms as similar to what he experienced in his lower limbs. It is inconsistent for Dr. Brawer to consider these upper body symptoms as relevant to a neuromuscular or neurological diagnosis, and to characterize these same symptoms as irrelevant when they appeared in Petitioner's lower body.

Dr. Brawer then ignored Petitioner's extremely high B6 levels in his second assertion that there were no other known potential causes of Petitioner's symptoms. Petitioner's treaters considered Petitioner's high B6 levels, his status as a CPII carrier, a differential Lyme disease diagnosis, fibromyalgia, and other possibilities as potential causes or co-factors in the development of Petitioner's symptoms. While Dr. Brawer may not believe these other possibilities are causal, he must articulate the basis for his belief for his argument to be persuasive. Lastly, Dr. Brawer relied on the temporal relationship between Petitioner's neurological symptoms and his vaccination. The appropriateness of this temporal relationship will be discussed later in this decision, but Dr. Brawer's decision not to characterize Petitioner's pre-vaccination symptoms as possibly neurological, while taking care to distinguish them from later symptoms strains the credibility of the entire argument. Dr. Brawer did not provide persuasive evidence to support Petitioner's claim that his neurological and neuromuscular injuries were caused by the flu vaccine.

Petitioner provided some evidence of an initial immune response immediately after vaccination, which Dr. Rubenstein used in support of his autoimmune neuropathy diagnosis. Although Dr. Donofrio did note that Petitioner did not report his initial fever to his treaters, Dr. Donofrio did not dispute the credibility of this account in Petitioner's affidavit and as reported to Dr. Rubenstein. Neither will I. Dr. Donofrio instead wrote that no neurologist found evidence of

an autoimmune process. Dr. Donofrio's conclusion assumes that evidence of autoimmunity is best detected by neurologists, that such evidence is obvious and unambiguous, and that a neurologist had the opportunity to detect any such evidence during the relevant time frame. While these may all be true, Dr. Donofrio did not adequately support these assertions during his testimony. Dr. Donofrio stated that autoimmunity is best detected by biomarkers in the blood, but he later noted that in order to relate an immune response to vaccination, the biomarkers would have to be detected within seven to ten days post vaccination. The fact that this testing was not done during this time period does not close the door on the argument. Dr. Donofrio testified that he would expect to see a fever the night of vaccination to indicate development of an autoimmune neuropathy. Petitioner provided evidence of such a fever, coupled with an ever-growing number of symptoms affecting everything from his muscle mass to his sense of touch. Some autoimmune diseases are difficult to identify, because many have an unknown etiology and present with a varied array of symptoms. Petitioner has presented preponderant evidence that he experienced symptoms in the days and weeks following his vaccine.

It is clear from the medical records, the expert reports and testimony, and the filed literature, that many of Petitioner's symptoms are not typically seen with small fiber neuropathy. In fact, Petitioner did not establish that many of these atypical symptoms, particularly his muscle atrophy and fatigue, are related of Petitioner's small fiber neuropathy. These two symptoms were reported by Petitioner pre vaccination.

Dr. Donofrio opined that Petitioner's sensory abnormalities could have preceded his vaccination, because neuropathy can be asymptomatic for an extended period. Dr. Donofrio was not, however, able to say that this was the case for Petitioner. Dr. Donofrio testified that he "didn't see a good thorough neurologic exam before the vaccination to base his answer." Tr. 180:17–18. Indeed, Dr. Donofrio conceded during that line of questioning that "there is no data" to substantiate the fact that Petitioner had a sensory neuropathy prior to vaccination. Tr. 180:23. Dr. Donofrio acknowledged that Petitioner's pre-vaccination complaints did not lead to neurologic testing by his physicians. However, the addition of Petitioner's post-vaccination symptoms ultimately led to the testing for and diagnosis of a neurologic disorder. Furthermore, the inability for several world-renown physicians to accurately diagnose Petitioner, despite his insistence that something was wrong is persuasive evidence that Petitioner's condition is rare and presented in an atypical fashion. Several of Petitioner's treaters believed that his vaccination could have played a role in the development of his neuropathy. Drs. Mercadante, Thomashow, Cudkowicz, Hirano, and Horowitz all noted Petitioner's vaccination as a possible cause or factor in the development of his neuromuscular condition. Although there was no consensus, it is compelling that multiple treaters, including neurologists, associated Petitioner's condition with his vaccine. Petitioner does not have to provide a step-by-step, line-by-line recipe for the development of his illness. A logical sequence of cause and effect will do. Given the totality of the evidence, I find Petitioner established, by a preponderance of the evidence, that his flu vaccine caused the development of his symptoms that were ultimately used to diagnose his small fiber neuropathy.

D. *Althen* Prong 3

Finally, Petitioner ultimately reported that he suffered from symptoms that could be associated with an autoimmune response, such as a fever, immediately following his vaccination. Petitioner further stated that he developed muscle weakness, and approximately one month later, he developed symptoms associated with neurological dysfunction to include numbness and foot pain while walking. Since his initial complaint, Petitioner consistently sought treatment for a growing list of symptoms until he was first diagnosed with a sensory neuropathy in March of 2012. Petitioner's treaters were not able to identify a cause for all of Petitioner's symptoms. However, his initial complaint approximately one-month post vaccination eventually lead to his diagnosis. A one-month onset falls within the appropriate timeframe for the development of a vaccine-induced autoimmune disease. I find that Petitioner has established a proximate temporal relationship between his flu vaccination and the development of his symptoms that ultimately lead to his neurological diagnosis.

E. Alternative Causation

The fact that Petitioner's physicians looked to other causes for his symptoms could support the assertion that they did not believe his vaccination was the cause of his condition. However, several of Petitioner's physicians noted in the record that at least some of his symptoms were, or could be, caused by vaccination. Additionally, treatments that were recommended based on alternative causation had mixed results. This suggests, for example, that Petitioner's B6 levels may have played a role in causing or exacerbating Petitioner's condition without being the sole cause. The evidence also strongly suggests that some of Petitioner's injuries may be related to some of his other known conditions, including his pre-vaccination chronic fatigue syndrome. Dr. Donofrio was unable to provide an opinion on the onset of Petitioner's neurologic symptoms. He would not say if Petitioner's condition developed pre or post vaccination. Respondent asserted that B6 toxicity or other factors were likely causes but concluded that small fiber neuropathy is largely idiopathic. Respondent also asserted that Petitioner's symptoms as reported in the medical records were not consistent with the appropriate timing of a vaccine-induced autoimmune injury. All of these assertions have been considered and do not provide a more likely than not alternative cause. I do not, therefore, find that Respondent established by a preponderant standard that Petitioner's other conditions (CFS, B6 toxicity, etc.), individually or together, are the sole substantial factor(s) of Petitioner's small fiber neuropathy. *See Lehrman v. Sec'y of Health & Human Servs.*, No 13-901V, 2018 WL 1788477 (Fed. Cl. Spec. Mstr. Mar. 19, 2018).

VII. Conclusion

Petitioner has put forth preponderant evidence that the influenza vaccine he received on November 18, 2011, was a substantial factor in his development of small fiber neuropathy; therefore, he is entitled to compensation with respect to that injury. This case shall proceed to damages.

IT IS SO ORDERED.

s/ Herbrina D. Sanders
Herbrina D. Sanders
Special Master